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MAY 1934

VOL. 90, No. 5

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# THE VETERINARY JOURNAL

## A MONTHLY REVIEW OF VETERINARY SCIENCE

Est. 1826

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# The Veterinary Journal.

MAY, 1934.

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GLADSTONE MAYALL, M.R.C.V.S.

MAY, 1934.

## Editorial

## HONOUR TO TWO WELL-KNOWN PROFESSIONAL MEN.

IT was not to be expected that so striking a personality as Lieut.-Colonel Dunlop Young could be allowed to retire from so prominent a position as that of Chief Meat Inspector to the Corporation of London without some mark of special favour and friendliness from those with whom his duties brought him in direct contact; and it was an honour to the veterinary profession, as well as to himself, when the Corporation Committee, under whose *ægis* he worked, entertained him to dinner and presented him with a suitably engraved set of silver plate as a token of the great esteem in which his services were held. That such mature experience as Colonel Dunlop Young had gained in other countries of the world as well as his own should be entirely lost was not to be thought of, and our readers will be glad to know that, as the Meat Adviser to several Colonial Governments, he will still be available to help with his advice any of his professional colleagues.

To Dr. Fred Bullock, LL.D., the ever-popular and courteous Secretary to the Council of the R.C.V.S., we extend congratulation on being decorated by the French Government, for services rendered, as " Chevalier " of the much-coveted French decoration of the "Légion d'Honneur"—the highest Order in France. For some years, and especially before the War, Dr. Bullock helped the French Government very much on certain Colonial matters in Northern Africa, and for this he was decorated with the Order of the Niftitan. Since that date Dr. Bullock has consistently done *liaison* work whenever opportunities offered, and the added Order of the Legion of Honour has been well deserved and well earned.

## RETURN OF THE INTERNATIONAL HORSE SHOW.

EVERY veterinary surgeon will welcome the reappearance of the International Horse Show after a brief interregnum during which it was feared that the effort to accomplish its return would not be successful.

As a spectacle the Londoner loves it, and the habitual frequenter of Olympia felt that something had definitely been missing during the past twelve months; whilst the veterinary surgeon, whose professional instincts unwittingly drew him to the ring, fully recognises the great part these functions and ceremonies play in the social and moral life of our country.

That the horse is not dead is shown by the fact that there are still nearly 800,000 of them left in Great Britain, and their healthy monetary value runs into millions of pounds. To keep so large a number—or even a high proportion of them—in health, is a national necessity, for the wealth of an owner depends on the health of the stock, and the health of the stock must depend on the scientifically trained veterinary surgeon.

With the present shortage of veterinary graduates there was never a better time for the young man to enter the profession, and the revival of the International Horse Show is a good sign of the times; whilst the fact that the evening of June 20th opens with a Ball at which H.R.H. the Duke of Gloucester (President of the Royal Veterinary College) has graciously promised to attend (the proceeds to go to help the Endowment Fund of the College), is indicative of the goodwill which exists between the College and its numerous helpers.

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## OUR NEXT SPECIAL NUMBER.

IN fulfilment of the plan which we have already placed before our readers, it is arranged to make the June issue of THE VETERINARY JOURNAL a special number devoted entirely to matters relating to dairy cattle, and it is very opportune that attention should be drawn to this subject at the present moment on account of the depression in the value of these animals in the open markets and stock sales of the country.

The breeder of stock—whether kept for store or reared for sale—is passing through a very critical time; and, indeed, one can comment truly upon the situation when one says that the stock breeder has had a minus return upon his outlay for some years, and has had increasing difficulty in keeping his head above water in consequence. To those of the veterinary profession whose livelihood is gained in country practice, this depression in farming has reacted very adversely, for the prosperity or otherwise of the breeder and owner of stock and his veterinary adviser must always go hand in hand. We, as well as our farmer clients, look to the authorities of the Ministry of Agriculture to set their house in such order that it shall again radiate joyous activity and place Great Britain once again in the limelight as the premier country of the world for pedigree stock of all kinds of agricultural animals; as we do not forget that when the farmer flourishes the veterinary surgeon flourishes also.

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# General Articles

## THE OSTEODYSTROPHIC DISEASES OF DOMESTICATED ANIMALS.

By SIR ARNOLD THEILER, K.C.M.G., Hon. Assoc. R.C.V.S.

### (3) The Osteodystrophia Fibrosa.

In human pathology the diseases " ostitis deformans " of Paget and " ostitis fibrosa " of Recklinghausen have been treated as allied diseases, which from the histogenetic point of view could be brought under one heading. Paget's disease was considered to be a localised one, affecting one or more bones, and Recklinghausen's a generalised form, spreading over the whole skeleton. Christeller introduced some sort of classification, distinguishing a porotic and a sclerotic form, the latter representing the recovery stages of the former. In each of them he again distinguished " hyperostotic " and " hypostotic " forms according to the prevalence of building up or resorptive processes, and subclassified further into juvenile and senile forms, according to the age of the patient. This classification was the result of investigations extended to the domesticated animals and applied descriptively to horses, goats, dogs, pigs and certain cases from zoological gardens, particularly in apes. Recently, however, objections have been raised against the identification of Paget's disease with that of Recklinghausen, and it has been urged that there are sufficient differences to warrant the conclusion that they are really two distinct morbid entities.

The changes which characterise ostitis deformans and ostitis fibrosa, and which must guide us in differentiating from other diseases, such as rachitis and osteomalacia, consist in an excessively accelerated and intensive process of resorption and rebuilding of the tela ossea. It would appear that the process is in principle identical in Paget's ostitis deformans and in Recklinghausen's ostitis fibrosa, and is manifested by abnormally increased activity of multinuclear osteoclasts, and the presence of much fibrous tissue which has assumed an osteogenetic function. The distinguishing features between the two are said to be the characteristic building up process of the tela ossea in Paget's disease resulting in certain peculiar histological changes called " mosaic structures " by Schmorl, and now considered to be specific. These " mosaic structures " are explained as due to differences in the process of resorption and rebuilding. The resorptive process in Recklinghausen's disease extends over large regions of the old bone tissue, which are refilled with new bone, whereas in Paget's disease smaller circumscribed portions are removed and replaced piecemeal with the new tissue. Hence in Recklinghausen's disease the deposition of new bone is more uniform, and the resulting structure resembles the normal structure more closely than in Paget's

disease, where the new structure is extremely irregular, with numerous demarcation or cement lines running in various directions and crowded into a small area, and so producing the peculiar mosaic picture.

Concerning the nomenclature of both affections, it has been realised that the term ostitis does not really fit the pathological process, and the term " osteodystrophia " is now used. In this paper I shall therefore refer to Paget's disease as " osteodystrophia deformans," and Recklinghausen's as " osteodystrophia fibrosa."

I have already drawn attention to the occurrence of osteodystrophia fibrosa in domesticated animals, and pointed out that in the past it has often been described as rickets and osteomalacia, or as osteoporosis. Joest and Zumpe were the first to use the term ostitis fibrosa for a disease in horses which they realised did not correspond to osteomalacia as defined by medical pathologists. They based their diagnosis on the examination of several bones of the skeletons, which clearly revealed the fact that they were dealing with a distinctly different disease. But even before this, other observers, particularly Elliot, in Hawai ; Robertson, in Cape Colony ; Mohler, in North America ; Carougeau, in Madagascar ; and myself in the Transvaal, had occasion to deal with the same disease, and had expressed the opinion that it could not be identified with osteomalacia. These authors used the term osteoporosis, or when describing it as equine osteomalacia did so under a reservation which made it clear that the disease was not identical with osteomalacia in other animals. In my publication of 1905, I referred to microscopical examinations of mine which showed the presence of large amounts of fibrotic tissue in the tumour-like changes of the maxilla, and compared the disease with an ostitis rarificans. Sturgess, dealing with the same disease in Ceylon under the local name of osteoporosis, makes it clear in his later references that he understood it to be ostitis fibrosa. The contribution of Sturgess and Crawford, published in May, 1928, provides data which throw considerable light on the pathogenesis of the disease, and is in line with the Japanese work of Niimi and Aoki, who in 1927 produced the disease artificially by a restricted dietary régime. Although the Japanese workers use the term osteomalacia, it is clear from their description and illustrations that the disease corresponds to what we now call osteodystrophia fibrosa. The more recent publications of Kintner and Holt, in September, 1932, also use the term equine osteomalacia, and the authors seem satisfied with their diagnosis without, however, giving full consideration to other possibilities. Fortunately their article contains good reproductions of clinical and micro-pathological changes, so that the reader is free to make his own interpretation, and I personally would have no hesitation in substituting the term osteodystrophia fibrosa for their osteomalacia.

My more recent histological studies have been carried out on material kindly supplied by Sturgess and Crawford from Ceylon, whose skeletal specimens and photographs of clinical cases leave no doubt about the correct identification of the disease, and on material collected from South Africa, where in earlier days

I had occasion to observe the disease myself. I am publishing the pathological details elsewhere, but propose here to refer to the principal changes found in the material at my disposal.

With regard to distribution, osteodystrophia fibrosa of equines affecting horses, asses and their hybrids occurs sporadically, and in enzootic or even epizootic form, all over the world. The sporadic form is often referred to as " bran disease " in different countries of Europe, a name which, as will be shown later, is not without significance. In English-speaking territories it is often referred to as " big head " and " bone disease." The French generally use the name " cachexie osseuse." The sporadic form has probably been seen in almost all countries at one time or another, although, curiously enough, the occurrence as enzootic or epizootic has been described mostly in countries of tropical or sub-tropical climate—a fact which until recently has helped to obscure the ætiology of the disease. It has been reported from Ceylon, Madagascar and the Philippine Islands. At one time it was very prevalent in South Africa, and was also described in the Congo and in the Cameroons. In earlier days its presence was also reported from various states of North America, as well as from South America (Brazil). On the occasion of my visit to Australia I was informed by Bordeau that osteodystrophia fibrosa was seen by him under different dietetic conditions and circumstances which suggest certain hereditary influences. It also occurred at one time in Caledonia, Fiji, Cochin China, Southern China, India and Java.

Although the disease is recorded in horses, donkeys and mules, it is more often discussed in the economically more important horse. It appears to attack horses at all ages after weaning, the foal apparently remaining free from the disease until its life becomes independent of its mother—an observation parallel with the case of the calf in bovine osteomalacia. Young horses seem to suffer more than old ones, the most susceptible age being stated as below seven years. Horses doing heavy work and fast horses in training are particularly subject. Where the disease occurs in enzootic form it is commonly stated that imported horses are more susceptible than native horses; but this was certainly not the case in South Africa, where imported and locally-bred animals of the same class were equally affected. Some observers reported that an enzootic might arise independently of climatic and soil conditions, while others reported that horses with a free run on soils rich in lime did not contract the disease, and that affected horses frequently recovered when shifted to such soils. Elliot, in Hawai, a country of volcanic origin, found the disease in a portion of the island with an unusually high rainfall, but not in the dry districts. According to Carougeau the disease was not known in Madagascar before the conquest in 1895, after which a large number of horses, mules and asses were imported, and then cases occurred. At one time it became the most serious disease of equines in Madagascar, and was regarded as the inevitable end of all imported horses, as they were rendered useless in the course of three to four years.

In South Africa the disease was first observed by Hutcheon in Port Elizabeth during the years 1885–89 amongst the race horses of one particular stable, and its subsequent history in the sub-continent is interesting. The first cases occurred in a colt and two fillies from the same sire, so that Hutcheon at first conjectured a family predisposition. But subsequently the disease appeared in unrelated horses introduced into the same stable, and was most pronounced in young horses in training. It was not known on the farm on which the horses had been bred. Of the cases recorded, the majority occurred in three-year-old animals. Thereafter less was heard of the disease until a serious outbreak occurred in 1898 at the military camp at Wijnberg amongst a troop of stabled horses imported from the Argentine, where they had been reared on pasture. Nearly all also suffered more or less from biliary fever (Nuttaliosis), but the first indication of a serious situation came with military manœuvres, in which a large number of the horses became lame: some apparently from splints, some from spavin and ringbone, but others from no obvious cause. When examined by Hutcheon, about eighty horses were suffering from various degrees of lameness in various forms. Some were unable to rise without assistance, and some were even unable to stand when lifted. A careful examination revealed the presence of the disease previously encountered at Port Elizabeth and then called "osteoporosis." The disease was also observed on a considerable scale by W. Robertson in Cape Town up to about 1904, and an interesting fact was brought to his notice by a Malay livery-stable keeper in a suburban village. This man stated that the disease had first occurred eleven years before in a broken down racing pony from Port Elizabeth, owned by a coloured man, in whose stable it died. The year after a Cape horse kept in the same stable contracted the disease and died, and since then, the Malay added, "the disease has always been with us." Similar observations were also brought to my own notice in the Transvaal, and it looked very much as if the spread of the disease could generally be traced to the introduction of a diseased horse into a healthy stable. I carried out certain experiments in which a diseased horse was stabled between healthy horses, but although I failed to transmit the disorder experimentally, I could not at that time get rid of the suspicion that we were dealing with a transmissable disease. Hutcheon had stated that after the gold-fields of the Witwatersrand in 1886 were opened up, and Johannesburg became the principal racing centre in South Africa, osteoporosis became prevalent. Race horses from the other colonies were sent there, but owners were afraid to keep them in the town for any length of time for fear of contracting osteoporosis. The disease then appeared to spread, and became equally prevalent in all classes of horses in the town. In 1893, I had occasion to observe the disease myself in Johannesburg amongst the horses of the mounted police and of private owners, and also amongst horses of the local tramway stables, where it played considerable havoc. After the Anglo-Boer war an alarming outbreak occurred in the military depot of Middelburg, Cape Province, in 1904, amongst horses, mules and donkeys, and from there I received material for experimental investigations. The bones of some of the animals were given to Ingle for chemical analyses. In the same

year Robertson observed no fewer than 156 mostly isolated outbreaks, and stated that the disease had evolved from a " pathological curiosity to a serious danger." No class of horses was spared. After that the disease gradually diminished, and finally became a rarity in all four provinces of the Union. The last cases seen by me were isolated ones: a horse in July, 1924, and a donkey in 1925, from both of which I was able to obtain post-mortem material for microscopic examination. The younger generation of veterinarians in South Africa have not seen the disease. It seems to have disappeared completely, and yet formerly it occurred under all possible conditions of stable hygiene, and no specific cause could be incriminated. As we shall see later, all the modern evidence goes to show that the disease is of dietary origin, but I have quoted the South African experience to point out that a quarter of a century ago ordinary logical interpretation of the observations would have suggested a disease transmitted by a vector, associated with an affected horse. The truth has only been arrived at by successful experimental production of the disease, although now that the real ætiology is fairly clear, it is no longer difficult to find an explanation for the earlier " apparent transmission." The present-day ætiological explanation treats the disease as caused by low calcium in the diet, associated with excess phosphorus; but before discussing the evidence for this, the disease itself may be described.

## Clinical.

Equine osteodystrophia fibrosa is a chronic disease without fever, which usually runs a course of several months, but may last a few years. At first it often passes unrecognised, but in countries in which it is known as an enzootic it is suspected quite early from changes in gait. The animal may stumble more than usual, or may be slightly stiff; it may spend more of its time lying down, become lazy and tire soon when put to work. As time goes on these symptoms become more acute, walking seems to become painful, and lameness may appear in one or more legs. Sometimes the seat of lameness can be traced, but very often not ; it may be a shifting lameness, and be put down to rheumatism. In the incipient stages of the disease the condition of the animal does not suffer and the appetite remains normal. Later the animal loses condition, develops a " tucked up " appearance, and the costo-abdominal groove becomes very prominent. In the later stages the animal is anæmic and cachectic. The pathognomonic feature is swelling of the jaws, first recognised as a thickening of the alveolar margin of the mandibula, but later spreading over the whole bone. The enlargement is most marked in the region of the molar teeth, extending backwards and forwards, so that the intermandibular space is much reduced. Simultaneously with the enlargement of the mandibula, swelling begins in the same region of the maxilla, and gradually encroaches on the lacrimal and zygomatic bone. In severe cases the swelling may extend to the nasal and frontal bones. When the palate is involved, as is commonly the case, the nasal cavity is reduced, and breathing becomes difficult. In advanced cases the hard compacta is replaced by a soft tissue, yielding to pressure and interfering with mastication,

XUM

so that the animals have difficulty in chewing food, and often drop the bolus. The buccal cavity becomes constricted, the point of the tongue rests between the incisors, sores may appear and the breath become fœtid. With increasing swelling of the bones the head becomes monstrous, justifying the English term "big head." In very bad cases deformations in other parts of the skeleton can also be found, particularly in the shoulder. The scapula may thicken and curve, causing displacement of the shoulder joints, which then project forwards, creating a sunken appearance of the sternum. The vertebral column may curve downwards (lordosis), and the costal arc flatten, conferring a peculiar aspect characteristic of the disease.

A very typical phenomenon of equine osteodystrophia fibrosa is the frequency of bone fractures and detachment of ligaments. The ribs are particularly fragile, and in severe cases simple pressure with the hand is sufficient to break them. The lumbar vertebræ sometimes fracture and cause paraplegia. The long bones of the legs may snap ; fracture of the sesamoids occurs fairly frequently, sometimes with no other apparent cause than the turning of the horse in the stall. Such a fracture may happen in a race horse during an actual race, without any premonitory clinical symptoms, as first indication of the serious skeletal disturbance. Although exostoses are found in diseased horses, they are by no means so conspicuous as to impress the examiner, and there is no deformation of the extremities as in bovine osteomalacia. If an animal happens to be spared from fractures it becomes cachectic as the disease progresses, finally lies down, and may be unable to rise again. At this stage any disturbance may cause several fractures at once, and place the animal beyond recovery. Recovery may occur in early recognised cases shifted to other areas or subjected to treatment. Such cases have been reported by several observers, including Carougeau, Sturgess, Kintner and Holt, and others.

The description I have given is a general one, but special attention should be drawn to the fact that the swellings of the maxilla and mandibula do not invariably occur. Robertson quotes nine cases out of 150 in which they were absent. Similar experiences are reported from other countries. The tumour-like changes in the maxilla and mandibula indicate a far-advanced stage of the disease. In the more recent observations, attention has been drawn to the fact that the urine is often acid owing to preponderance of acid phosphates, but this is not diagnostic, and may vary in one and the same animal from time to time. The latest publication by Kintner and Holt provides valuable data on the blood findings in the case of thirty-six sick horses and sixty-nine normal horses (recently imported into the Philippines), particularly in respect of calcium and phosphorus. On the average the blood calcium was reduced about 9 per cent. and the inorganic phosphorus raised about 20 per cent. in the disease. The authors use the "ion product" to compare their data with the blood findings in rickets, and show that on this basis there is no similarity; in the disease of horses the "ion product" was higher in the normal than in the affected animals. The Japanese authors,

1882

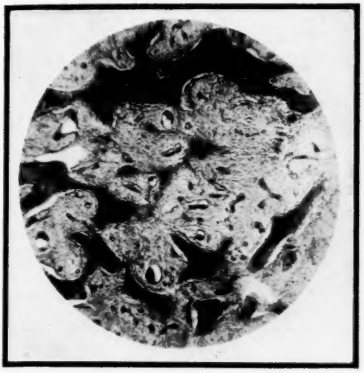

FIG. 19.—Arab stallion. Maxilla. The greatest portion of the old compacta has been removed, and its place is occupied by an abnormal fibrous tissue, in which the production of fibrous bone trabeculæ is taking place. Mag. 39 ×.

FIG. 20.—Arab stallion. Intermaxillary bone. Removal of the old compacta through the resorptive activity of a fibrous tissue, in which fibrous bone trabeculæ are formed. Mag. 39 ×.

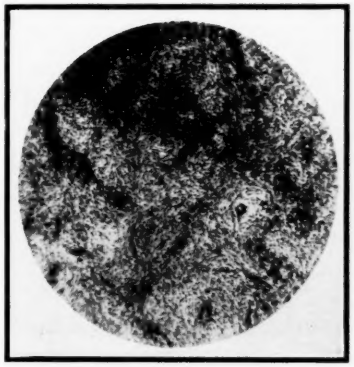

FIG. 21.—Horse 4,150. Maxilla. Not decalcified. A fibrous tissue, simulating a fibroma, replaces the former tela ossea. Mag. 63 ×.

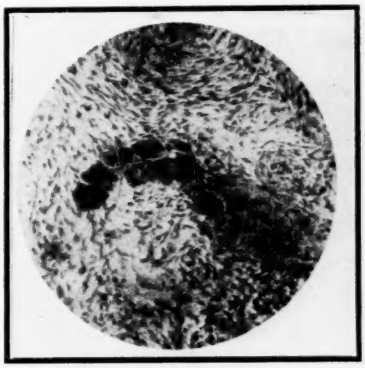

FIG. 22.—Horse 4,150. Maxilla. Not decalcified. A portion of the fibroma-like tissue, which includes osteoclasts. Mag. 152×.

1886

FIG. 23.—Horse 4,150. Third rib. The greatest portion of the former spongiosa has been removed, and is replaced by new fibrous tissue producing woven trabeculæ. The lymphoid marrow undergoes atrophia. Mag. 39 ×.

FIG. 24.—Horse 4,150. Thirteenth rib. There is a subperiosteal osteophytic formation of fibrous bones, the trabeculæ of which are radially arranged to the longitudinal axis of the rib. Mag. 39 ×.

FIG 25.—Horse 4,150. First thoracic vertebra. Epiphyseal cartilage. The subchondral zone has been completely removed and replaced by a fibrous tissue, in which extensive formation of new woven bone trabeculæ has taken place. The provisional calcification zone of the cartilage is calcified. Mag. 39 ×.

FIG. 26.—Horse 4,150. Pelvis. The osteoclastic process has invaded the compacta. The lymphoid marrow is replaced, or is being replaced, by fibrous tissue. In some places there is marked activity of osteoclasts. Mag. 39 ×.

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FIG. 27.—Horse 4,150. Femur. The tela ossea of the subarticular zone has been removed and replaced by new fibrous bone The process of removal is still progressing, and marked osteoclastic activity can be recognised in a rather large groove. Mag. 39 ×.

FIG. 28.—Arab stallion. Femur. A typical hernia of the articular cartilage. The photo shows how the calcification zone has been pushed aside, and the cartilage entered the subarticular groove, produced by the osteoclastic process Mag 39 ×.

Niimi and Aoki, reported decreased blood calcium and increased urinary phosphate in their experimentally-produced cases. Since these were produced by exclusive or predominant cereal feeding, and cereals contain a large excess of phosphoric oxides over basic oxides, the urine of the horses would, of course, be acid.

**Morbid Anatomy.**

The striking post-mortem findings in an advanced case of osteodystrophia fibrosa in a horse is the porotic condition of the whole skeleton, a change which is so striking that it is only natural that the disease should have been referred to as osteoporosis. The condition is best observed in macerated bones, and can easily be recorded by a comparison of the weight of such bones with that of the same bones of healthy horses similarly macerated. Such comparisons have been made by several investigators, always with the same result. This difference in weight is very remarkable, amounting to 50 per cent. or more, or to a loss in weight of 41 to 70 per cent. in the case of the osteodystrophic skeleton. The porotic changes are well shown by X-ray photographs; and radiography, as practised by Kintner and Holt, is a useful method of diagnosis. The most conspicuous changes are found in the swollen parts of the head, in the maxilla and mandibula, the affected portions presenting the appearance of tumours in which the molars are embedded with their tables levelled to the alveolar margin. The normal structure of the bone is entirely gone, and its place is taken by a softer tissue quite differently built. Microscopical examination of an advanced case shows that the old compacta has been replaced by a fibro-cellular tissue, which also fills the spaces of the former medullary cavities. This soft tissue usually includes bone trabeculæ, or fragments of trabeculæ, more or less regularly arranged in some places, but mostly scattered about in irregular fashion. In some places there may be no trabeculæ at all. The trabeculæ are produced by the fibro-cellular tissue, and can be recognised in various forms of transition, from a simple grouping of osteoblasts depositing osteoid to a definite trabecula, consisting of more or less calcified osteoid. The younger the trabecula appears to be, the more marked is the osteoid tissue, with its ensheathing envelope made up of one or more rows of osteoblasts. In some places the structure resembles that of a fibroma, and there are even parts which suggest a sarcoma. Where an agglomeration of osteoclasts simultaneously occurs, the picture resembles somewhat that of a giant-cell sarcoma. In the human patient the corresponding structure is compared to an epulis, and referred to by some authorities as the "intra osseus epulis." A fair amount of dark or yellowish pigment is usually present along with some extravasated blood. In portions adjacent to this structure the osteoblasts and osteoclasts are less numerous, the tissue has a more fibrous structure, the trabeculæ are longer and more systematically arranged, as if according to some structural design.

In less advanced cases, where the tumour-like tissue is not pronounced, the evolution of the morbid changes can be followed by examining a serious of sections from different localities. It then becomes evident that the old compacta, in which

the outer regions of mixed systems and the inner regions of Haversian tubes can still be recognised, is broken down by numerous multinuclear osteoclasts, which rapidly remove the bone tissue from within and without in most irregular fashion. The removed portions are replaced by a new tissue deposited by osteoblasts lining the lacunæ. This new tissue sometimes shows distinct osteoid margins. The new tela ossea may be again removed and replaced. Trabeculæ reappear on the outside of the old compacta, arising in the periosteum and running radially to the surface of the bone. They form an osteophyte, which ensheathes the mandibular and maxillar bones, and the osteophytic growth is partly responsible for the increase in volume of both bones. On the inner side of the former compacta the fibro-cellular marrow produces similar trabeculæ, which occupy the region of the former spongiosa. At certain stages a cross section through the mandibula may reveal three different structures : an outer osteophytic zone, a central portion of old demolished compacta, and an inner zone composed of fibro-cellular trabeculæ. The breaking down of the old compacta proceeds steadily until finally it is completely removed. The destructive process may ultimtely invade all the bones of the skull. In the nasal, frontal, temporal and parietal bones the tabulæ interna and externa, together with the spongiosa of the diplœ, are gradually replaced by an altogether different trabecular structure. Similar changes are found in the flat bones of the skeleton: the ribs, scapula and pelvis, which are all soft and easily cut. Many of the affected bones show increased volume, particularly noticeable in the distant portions of the ribs and certain parts of the pelvis, which are round or oval in cross sections. The changes described for the mandibula are also observed here : an osteophytic growth envelopes the compacta, which along with the spongiosa, is replaced by a new trabecular architecture. Peripheral trabeculæ are placed perpendicularly to the bone surface, while trabeculæ in the centre of the bone are arranged in whorls. In some bones the old compacta appears to be first split longitudinally into leaves by " dissecting resorption," and then broken down further, removed, and simultaneously replaced. The final architecture has no resemblance to the original one, but the shape of the bone as a whole is approximately maintained.

A constant lesion to which all observers have drawn attention is the presence of local defects on the cartilaginous surfaces of the joints of the proximal articulations of the extremities, especially well marked in the patella They are commonly, but erroneously, described as ulcers. As a rule their margins are irregular, and they originate through the protrusion of a portion of the cartilage, like a hernia, into a cavity previously formed in the adjacent subarticular bony tissue by osteoclastic activity. The surface of the sunken cartilage becomes necrotic and peels off in thin layers, so creating a localised defect. The subarticular osseous zone bordering the calcified cartilage is a region in which far-reaching changes occur. This zone is removed by osteoclasts, which cut away the original tissue and attack portions of the calcified cartilage, thinning it down and rendering it conspicuously narrow. Removal may be so complete that practically nothing of the old structure remains, and replacement usually occurs by osteoblasts lining

the grooves in regular order, and filling the gaps by depositing osseous laminæ. Larger holes are also filled with bone tissue produced by fibro-cellular marrow, so that the new structure may be developed from different centres, the various portions finally coalescing and attempting to make good the destruction of original bone. The new zone differs considerably from the one it replaces, and its behaviour towards stains shows it up at once. In bones semi-decalcified for section it stains more lightly, indicating that the new tissue was itself uncalcified, or only imperfectly mineralised. The width of this transformed subarticular zone varies in different articulations. Similar changes are found in the regions adjacent to the intervertebral cartilages, portion of which bulge into cavities excavated by the destructive process. The compact tela ossea of places where sinews are attached undergoes similar changes. Very conspicuous alterations occur in the subchondral zones of the functional cartilages, particularly well marked in the vertebræ and ribs. The cartilage of conjugation generally appears normal, but the zone of provisional calcification is narrow and sometimes excavated. The regular normal arrangement of primary canals, intended to remove hypertrophied cartilage cells and to deposit lamellar bone on the septa, and so form the first osseous pillars linking up with the spongiosa, is completely lacking. The endochondral process of bone formation has ceased operation, and the subchondral zone is filled with fibro-cellular marrow, which forms irregular trabeculæ, replacing former endochondral pillars. In some cases the functional cartilage is broken through, and trabeculæ of woven bone fill the gap and appear on the opposite side. Multicellular osteoclasts are observed removing the old tissue, and even newly-formed trabeculæ are sometimes included in the absorptive process. Newly formed trabeculæ are often uncalcified or incompletely calcified, show red margins and an enclosing envelope of osteoblasts. The resemblance to the conditions seen in rickets is so superficial that very little observation is required to differentiate the process in operation, and once realised, the changes in the two diseases cannot be mistaken.

Changes are also found in the spongiosa of all the bones which, generally speaking, show advanced atrophy. The medullary cavities of the long bones are extended longitudinally and centrifugally. Sometimes the osteoclasts split the trabeculæ longitudinally and sometimes laterally. Simultaneously with this disruption and removal, rebuilding and replacement goes on. Older trabeculæ form the support for the deposition of new lamellæ, and in some places new tissue is formed by the origination of trabeculæ from the fibro-cellular matrix. The spongiosa in the vertebræ is also rebuilt, the new structure generally following the old pattern. The longitudinal trabeculæ are split through their central axes, thinned down and replaced by new trabeculæ emerging from the fibro-cellular marrow. The spongiosa in the pelvis, scapula and sternovertebra all undergo similar changes.

A striking feature, pathognomonic for osteodystrophia fibrosa, is the presence of fibro-cellular and fibrous marrow in all bones. The more severe the case the

more there is of it, and in very advanced cases the hæmatoplastic tissue has almost entirely disappeared. Wherever the process of transformation is accelerated, the fibrous marrow is rich in spindle-shaped cells and osteoclasts, and fibrous elements dominate wherever the structure appears to be reaching some measure of finality. Where lymphoid and fibrous marrow are present simultaneously, the former is pushed into the centre of the medullary space, and the trabeculæ are shrouded with fibrillar tissue.

The description I have given is based upon observations on the horse, but the disease osteodystrophia fibrosa has, as already mentioned, been recorded in other domesticated animals. In the pig it is sometimes called "snuffles." Mutatis mutandis, the clinical symptoms in severe cases correspond to those in the horse. The swelling of the maxilla and mandibula constricts the air passages and renders breathing difficult. There are also distortions in the bones of the skeleton and thickenings of the chondrocostal regions. The osseous tissue in the jaws is replaced by a fibroma-like fibro-cellular mass, which includes osteoclasts and pigment. Similar changes are seen in the dog, which, however, may also show deformation of the bones of the extremities and the "rickety rosary" in the ribs.

The microscopical changes are also similar to those described for the horse, although in the dog, pig and goat the presence of cysts in the tumour-like tissue of the maxilla and mandibula is sometimes mentioned. In these animals the excessive resorptive and rebuilding processes are also found throughout the whole skeleton, so that the disease can be closely identified with the equine osteodystrophia fibrosa.

In the experiments of Marek and Wellmann, already discussed, rickets in pigs was sometimes complicated with osteodystrophia fibrosa, and an association of the two diseases was also observed in sporadic cases in goats. These authors, however, do not consider the two diseases as radically different, and draw attention to the fact that there are lesions in rickets which differ only in degree from lesions in osteodystrophia fibrosa, and that there seem to be transitions from one disease to the other. To this view I am unable to subscribe, since I consider that the two processes are distinct, and that the extreme forms of the two diseases are so different from a pathological point of view that they certainly should be considered as independent morbid units. I prefer to think that in some animals, notably the pig, the two diseases can occur simultaneously.

According to the classification of Christeller, the disease as described by me in the horse would come under the heading of the hyperostotic form, since the jaws and certain other bones show increase in volume. But it should be pointed out that the terminology is open to misinterpretation, and might suggest more bone, whereas in fact only increase in volume is meant. Lesions without hyperostosis are found in many horses, in which case the process would be called hypostotic porotic. They are always present at the beginning of the disease, before the tumour-like swellings of the maxilla and mandibula are found. The funda-

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mental process is always hypostotic. As already pointed out, the weight of the skeleton is finally reduced by half. Sclerotic processes have not come to my own notice. It is, however, possible that what has been described as Leontiasis osseum or osteismus by Kitt is, indeed, nothing else but a healed-out sclerotic osteodystrophia.

The bones of diseased animals, particularly of horses and mules, have been analysed by various workers. Ingle, in South Africa, comparing the metatarsus of diseased and of normal mules, found the former lower in ash, lime and phosphoric oxide. The Japanese and Philippine investigators record similar findings.

The equine material I have examined did not show the " mosaic pattern " of Schmorl, and the various authorities who have studied the disease in goats, pigs and dogs, give no description which could be interpreted as such. It would appear that in domesticated animals there is as yet no definite evidence that Paget's disease occurs, although it has been diagnosed in apes. The question now arises whether, from a comparative point of view, the generalised osteodystrophia fibrosa can be considered to be the same disease in the different species of animals and in man. In my opinion it probably can, since the fundamental changes appear to be the same, although the lesions in man differ in some respects from those in animals. In the human disease, in addition to the excessive resorption and rebuilding of bone, the presence of cysts and giant-cell tumours are mentioned as occurring in some cases. These tumours are known as the " brown tumours," and may be found in any part of the skeleton; they are often associated with cysts, which may be of considerable size. In the domesticated animals tumour-like thickenings have so far only been described in the maxilla and mandibula, and the tissue is not conspicuous by a brown colour, although it does show some macroscopic discolouration. Microscopically, however, the brown pigment is found, giant cells are present, and a fibroma- or sarcoma-like structure is quite typical in some cases. Although cysts have not been observed in the horse, they have been described in the tumour-like masses of the dog, pig and goat, differing mainly in size from those occurring in man.

Recent researches have drawn attention to changes in the parathyroid corpuscles in man. In Recklinghausen's osteodystrophia these bodies are frequently enlarged. In some cases the enlargements are interpreted as a simple hyperplasia, in other cases as adenomas. They are not found in all cases, and seldom in all four glands. The size of the growth is also variable. Human pathologists are inclined to connect these changes definitely with Recklinghausen's disease, assuming that hypertrophic parathyroids are never found in Paget's disease, and treat the absence as of differential diagnostic importance. In the explanation of the pathogenesis of osteodystrophia fibrosa in man, the parathyroid changes play an important rôle, since the epithelial bodies are considered to be regulatory centres for control of calcium metabolism.

So far there is little or no information on parathyroid lesions in domesticated animals. Certainly no examinations were made in the earlier outbreaks of the

equine disease in South Africa and Madagascar, since these occurred at a time when the possible significance of the parathyroids in this connection was not known. They are not mentioned by Crawford, nor by Niimi and Aoki, nor in the recent comprehensive publications of Kintner and Holt of 1932; nor in the publications of Christeller, Marek and other observers of osteodystrophia in the domesticated animals. When it was realised that the simultaneous occurrence of osteodystrophia fibrosa and parathyroid tumours in man was not mere coincidence, surgeons started to remove the tumours, and with a certain measure of success. Even the partial removal of what seemed to be normal parathyroids gave good results. This led to the conclusion that the parathyroid tumours were the primary cause, giving rise to defective calcium metabolism, which in turn caused the changes in the skeleton. The problem was then attacked experimentally. American investigators, Jaffe and others, injected standardised extract of parathyroids, parathormone Collip, into guinea pigs, and produced alterations in the skeleton considered to be identical with osteodystrophia fibrosa. They also succeeded in producing similar changes in the dog, but only when the injections were continued over a longer period. Similar results were obtained in rabbits in the pathological institute at Geneva.

Investigations on equine osteodystrophia fibrosa have taken two main directions, one school of thought considering the disease to be of infectious origin, and another associating it with a dietary régime. The former were influenced by its enzootic and epizootic appearance, and its spread after the introduction of diseased horses into a healthy stable; but they could not obtain any direct experimental evidence for their view, and so failed to prove their case. One observer, Conreur, considered the disease as it occurred in Brazil to be due to a heavy infection with cylicostomes, but this view has received no support. The supporters of the view that the disease was of dietary origin had tradition in their favour in the sense that " miller's disease," or " bran disease " of horses, must be considered as an osteodystrophia connected with one-sided feeding on cereals or bran, which are notoriously poor in lime and rich in phosphorus. In South Africa, Ingle pointed out that the prevailing ration of hay and maize was low in calcium but rich in phosphorus, and suggested that osteoporosis was due to an unduly wide ratio of $CaO : P_2O_5$. Although he had no experimental proof his views turned out to be well founded, and the later experiments in Ceylon, Japan and the Philippines may be regarded as establishing the true position. To Sturgess and Crawford, in Ceylon, belongs the credit of carrying out feeding experiments which are of fundamental importance, but which did not at the time receive the attention they deserved. Experiments on six ponies were carried out in 1926–27. For several years before Sturgess had observed the disease, particularly in two to four year old thoroughbred horses imported from Britain, and fed upon rations in which cereal products, particularly bran, figured largely. In certain stables addition of lime to the water was habitually made, and in these there was little or no disease. He and Crawford therefore subjected the " mineral ratio " view to direct test by selecting 10 lb. of bran as basal diet

and supplementing with Mauritius hay, calcium lactate and bone meal, so as to vary the CaO : $P_2O_5$ ratio. For the first nine months of the experiment the ratio was 1 : 5 in one experiment, 1 : 6.5 in another, and 1 : 18 in a third. With the exception of one pony on the 1 : 5 ratio, all showed clinical symptoms of the disease within nine months, the widest ratio operating most rapidly. The ratios 1 : 5 and 1 : 6.5 were altered to 1 : 14, except in the case of the pony which had remained healthy, and the experiment was continued for a further five months. The disease became intensified, and again the severity of symptoms corresponded to the divergence in the CaO : $P_2O_5$ ratio. Finally the diet of the remaining healthy pony on the 1 : 5 ratio was changed to 1 : 14. Within two months it showed clinical symptoms suggesting that it already had the disease in incipient form, and in less than three months it fractured its back and had to be destroyed. The total quantities of calcium, expressed as CaO, varied from 6.8 g. per day in the case of exclusive bran feeding, to 28.2 g. for the pony receiving a supplement of 2 oz. of bone meal. The quantity of phosphorus, expressed as $P_2O_5$, varied from 122 g. to 142 g., *i.e.*, was always in large excess. The pony which only slowly developed the disease on the 1 : 5 ratio, remaining clinically healthy for a long time, received the largest absolute amount of CaO. Its companion on the same ratio, but with somewhat less CaO, developed the disease within nine months, but only in a mild form. One pony was kept as control on a ration of oats, linseed, bran, chaff, grass and bone meal, containing 19 g. CaO and 37 g. $P_2O_5$, *i.e.*, with a ratio of rather less than 1 : 2. It remained healthy for fourteen months, when owing to a slight suspicion of incipient enlargement of the mandibula it was given an additional 2 oz. of bone meal for safety. This brought the total CaO up to 27.8 g., an amount equivalent to the maximum received by the two ponies on the 1 : 5 ratio. It remained perfectly healthy, in good condition, without increase in size of the mandibular, and with no sign of lameness. The earlier suspicion was treated as unfounded, and the pony was sold as sound after twenty months of observation.

Sturgess and Crawford also paid attention to vitamin factors, particularly vitamin C, but came to the conclusion they did not enter into the pathogenesis of the disease; indeed, from their data one can deduce that vitamin C is not necessary for the horse. They also considered the possibility of infection, but ruled it out as a possible agent in the experiment, and concluded that the ratio CaO : $P_2O_5$ was the important ætiological factor. Of special importance is the fact that the pony on 19 g. CaO and 37 g. $P_2O_5$ remained healthy for fourteen months, while another on about the same amount of CaO, but with somewhat more than twice as much $P_2O_5$ developed the disease within nine months. A general survey of their data makes it clear that the main cause of the disease is low calcium combined with high phorphorus, but also that the disease may appear even when the calcium is apparently adequate if at the same time the phosphorus is grossly preponderant. It is important to make a distinction between mineral deficiency in the absolute sense and mineral deficiency in the relative sense, and to remember that the disease may be expected either when the calcium is

too low or the phosphorus too high ; also to remember that the significance of ratio as distinct from absolute intake varies with different animals, and is much less important, as our South African work showed, for cattle. It is probably also less important for the human subject, as indicated by a survey of the mineral ratios in the dietary of certain African and Asiatic races.

About the same time as Sturgess, the Japanese were independently investigating the same disease, and Niimi and Aoki published their experimental data in 1927. In two experiments they fed four horses on barley only, and produced the disease in about five months, a rapidity explained by the fact that they not only had a highly adverse CaO : $P_2O_5$ ratio, but also a low absolute daily intake of calcium. The ratio in barley is about 1 : 13. In their first experiment the absolute daily intake works out about 2.1 g. CaO and 27.5 g. $P_2O_5$ for a ration adequate in starch value. In the second experiment, absolute intake was about 3.6 g. CaO and 47.4 g. $P_2O_5$. Two control horses received the same amount of barley (6 kg.), but with addition of 70 g. of calcium carbonate, and these remained healthy. The one-sided feeding with crushed or uncrushed barley, without hay, produced gastro-enteritis in the early stages of the experiment, more so in one horse than in the other, and signs of pica were also shown. The experiments of Niimi and Aoki lasted from 118 to 230 days, and these relatively short periods, together with the fact that fairly old horses were used for the experiments, account for the rather mild form of the disease. But there is no doubt about the diagnosis. The symptoms described and the morbid changes were typical of osteodystrophia fibrosa—swelling of maxillar and lacrymal regions, stiffness, lameness, weakness and fracturing of ribs. Chemical analysis of rib bones of experimental and control animals supported their diagnosis—reduced mineral content of the diseased bones. In comparing the data from the Japanese experiments with those from Ceylon, it may be noted that the lowest daily intake of CaO in the latter was 6.8 g., as against 2.1 g. to 3.6 g. in the former. It is safe to assume that 2.1 g. CaO was below the absolute minimum calcium requirements of the animals concerned, and that the Japanese horses would probably have developed an absolute calcium deficiency disease even if the CaO : $P_2O_5$ ratio had not been so adverse as it was ; this may account for the very rapid development of clinical symptoms in the comparatively old horses.

The investigations of Kintner and Holt in the Philippine Islands allow us to pursue the question of mineral ratio still further. A marked increase in the incidence of the disease occurred amongst the Army horses about 1924, one year after the ration had been changed from one containing imported foodstuffs to a cheaper one made up of native produce. Chemical analysis of the new ration showed a CaO : $P_2O_5$ ration of 1 : 2.9. The influence of minerals and vitamins was tested by adding 300 c.c. of cod-liver oil, containing vitamins A and D, to the daily ration of two horses, and 10 c.c. viosterol, containing vitamin D only, to the ration of one horse. The daily ration itself contained 14.2 g. CaO and 33.4 g. $P_2O_5$, *i.e.*, showed a ratio of 1 : 2.4. On later radiographic examina-

tion one of the two horses receiving cod-liver oil showed some improvement, but the viosterol appeared to be without effect, and it was concluded that the D factor was not ætiologically significant. The inclusion of green native grass in a ration containing 18.7 g. CaO and 45 g. $P_2O_5$ in one case, and 13.6 g. CaO and 31.6 g. $P_2O_5$ in another, failed to arrest the development of the disease. In a further experiment yellow maize, supposed to be rich in vitamin A, was included in a ration with a CaO : $P_2O_5$ ratio of 1 : 2.5, and sufficient ground limestone added to adjust this to 1 : 0.9. Of the six animals on this latter ratio, all improved within six months. The control horse on the 1 : 2.5 ratio showed no improvement. In a second series of nine animals on a similar ration without limestone, and a CaO : $P_2O_5$ ratio of 1 : 3 (14.2 g. CaO and 42 g. $P_2O_5$), the disease became progressively worse in eight cases. In a further experiment three groups of five animals were differentially treated. Group A received calcium chloride sufficient to bring the CaO : $P_2O_5$ ratio to 1 : 1.5 or 1 : 1.4. Group B received ground limestone sufficient to bring the ratio to about 1 : 1 (34 g. CaO, 36 g. $P_2O_5$), and group C received calcium phosphate and bran sufficient to bring the CaO to the same figure as group B (34 g.), but to raise the $P_2O_5$ to 85 g., and the ratio to 1 : 2.5. All the horses in group A and B improved, and radiographic examination showed that the disease had been arrested; but in group C, three out of the five animals became progressively worse. The interest of this experiment is that the calcium content was approximately the same in each group and well above minimum requirements, so that the behaviour of group C can be attributed to excess phosphorus or faulty CaO : $P_2O_5$, as distinct from deficiency of calcium. In another comparison the ration, including the calcium in the drinking water, of thirty-one horses recently imported from U.S.A., and sent to the military post at Fort Stotsenburg, contained 18.5 g. CaO and 53 g. $P_2O_5$, *i.e.*, a ratio of 1 : 2.9. The ration of 25 horses at Nichols Field, another military post, worked out at 15.7 g. CaO and 29.1 g. $P_2O_5$. Despite the lower calcium no cases of the disease occurred at the latter post, while three horses (10 per cent.) developed the disease at Fort Stotsenburg.

The records from July, 1930, to July, 1931, for the various Army posts revealed an interesting point in connection with the drinking water. The rations of the horses were approximately uniform, but at Fort Mills the water contained about 125 parts of calcium per million, whereas at Fort Stotsenburg it contained only 9.7 parts per million. No cases were reported from the former, but the incidence was about 10 per cent. at the latter. Calculating from analyses of the ration, and allowing an average consumption of 6.5 gallons of water per animal, the CaO : $P_2O_5$ ratios worked out at 1 : 2.3 and 1 : 2.9 respectively. When the general ratio was altered in 1931 to about 1 : 1 by a supplement of finely-ground limestone, there was a rapid and striking improvement in the records. Kintner and Holt therefore conclude, from their own work in the Philippines, and from a general study of the literature from other countries, that there is no evidence to show that the disease under discussion is due to infection, heredity, disturbances of internal secretion or vitamin deficiencies, but can be solely ascribed to mineral imbalance.

They define the critical ratio CaO : $P_2O_5$ fairly closely by showing that at 1 : 2.9 horses generally developed the disease. At 1 : 1.9 it did not develop in a nine months' period of observation. Feeding experiments showed that the disease was arrested on a ratio of 1 : 0.9, 1 : 1, and 1 : 1.4, and that it became progressively worse on a ratio of 1 : 2.4, 1 : 2.5, and 1 : 3.

Reviewing the observations from Japan, Ceylon and the Philippines, we see from the data of Niimi and Aoki that the disease can appear very rapidly (five months) even in fairly old animals, when the absolute calcium intake is very low (2.1 to 3.6 g. CaO), and the ratio CaO : $P_2O_5$ very wide (1 : 13) ; from the data of Sturgess and Crawford we see that even when the calcium is adequate (26 g. CaO) the disease can be produced in healthy young horses in about a year with a CaO : $P_2O_5$ ratio of 1 : 5 ; from the data of Kintner and Holt we see that the critical ratio for arrest or progress of the disease is even narrower, roughly between 1 : 2 and 1 : 3. To be safe, it should be nearer 1 : 1 than 1 : 2.

The Philippine observations are also of special interest in that the course ot the enzootic was not unlike that of the early South African outbreaks, which at the time suggested a disease of infectious origin, but which can now be explained on the basis of the later experimental work on mineral imbalance. In the same way it is now possible to trace back the history of enzootic outbreaks in several countries where they were formerly regarded as associated with infection, and show that the nutritional factor was probably operating all the time. For example, the South African outbreak in Wijnberg from September, 1898, to January, 1899, occurred amongst military horses under the supervision of Captain Lane, and although the mineral content of the diet is not precisely known, the Army ration itself was fortunately recorded in Hutcheon's report for 1904. This consisted of 10 lb. of locally grown oat hay, 7 lb. of maize, 1 lb. of bran, and ½ oz. of salt. If we assume the low values for calcium recorded for some South African oat hays, and assume average values for phosphorus, the daily quantity of CaO might well work out about 10 g., and of $P_2O_5$ not much below 50 g. This would give a CaO : $P_2O_5$ ratio of 1 : 5, a combination upon which osteodystrophia fibrosa could develop with considerable rapidity. In January, 1899, Captain Lane changed the ration to alfalfa hay, 8 lb., green forage, 4 to 6 lb., oats, 5 to 6 lb., bran, 2 lb., and bone meal, 1.5 oz. Assuming average figures for mineral content, the CaO would amount to about 69 g., and the $P_2O_5$ to about 62 g. In addition, unslaked lime was added to the drinking water in small quantities, so that the CaO : $P_2O_5$ ratio would be well within 1 : 1, say 1 : 0.9, the figure upon which Kintner and Holt reported rapid recovery. Captain Lane's records show improvement, no matter how far the disease was advanced, often noticeable in a few weeks. With the exception of about a dozen bad cases, out of about 400 horses, all the animals became workable. Nevertheless, Hutcheon, who reports the history of the outbreak, was not convinced that the disease was of direct dietary origin. He considered that the improved ration was a contributory cause in recovery, but that the history of the outbreak pointed to

the introduction of an originating cause from the outside. In the light of recent knowledge we can dismiss the hypothetical originating cause, and explain the whole incident on a dietary basis. The same applies to my own observations in the Transvaal. Although coincidence, that curious disturber of deductive reasoning, always provided a diseased horse to start up the outbreaks, the newer knowledge derived by the inductive process suggests that it was only a red herring drawn across the trail. In most cases the ration of the stabled horses consisted of oat forage cut just before ripening, in which the $P_2O_5$ may considerably exceed the CaO, and maize in which the $P_2O_5$ is high and the CaO negligibly low. The heavier the maize feeding, the more divergent became the CaO : $P_2O_5$ ratio, so that the Philippine ratio of 1 : 3 could easily have been present in all cases, and mineral imbalance have been the sole cause of the disease. What to us in the early days seemed puzzling, *i.e.*, the fact that the disease was not met with in pasture horses, finds an easy explanation. South African pastures tend to be deficient in phosphorus rather than calcium; and even when calcium is low, as judged by European standards, as is often the case, the phosphorus is usually still lower. Kintner and Holt's critical CaO : $P_2O_5$ ratio is never reached, and hence the disease was unknown in horses running on the veld. The fact that the disease was rife thirty years ago, but disappeared completely before its ætiology had been discovered, is a little more difficult to explain. The foodstuffs have not changed their mineral content, and we can therefore only assume that the tradition of feeding stabled horses must have changed unnoticed. But perhaps the increased use of a variety of cultivated crops, including lucerne and teff, may be responsible.

Of peculiar interest in all this work on osteodystrophia fibrosa of the horse is the fact that the adverse CaO : $P_2O_5$ balance, which produces the disease, has no apparent ill effect on cattle : in so far, that is, as the matter has been experimentally tested. In one of our own experiments, recorded in the paper already referred to, two young heifers increased in weight from a few hundred pounds to over a thousand pounds in perfect health on a ration in which bran was a liberal component, and which showed a mineral ratio of about 1 : 3. The same apparently applies to pigs, since in the experiments of Marek and Wellmann a wide ratio was not always accompanied by the appearance of osteodystrophia fibrosa. In their experiments, already discussed, rickets of young pigs was sometimes complicated by osteofibrosis. On examining the CaO : $P_2O_5$ ratios of the relevant rations we find that they vary from 1 : 1.24 to 1 : 10.57. In four cases the ratio was wider, and in four cases narrower than the ratio which Kintner and Holt specify for the occurrence of the disease in horses. It would therefore appear that ratio as such is not the dominant factor in the ætiology of the disease in pigs, although further work is required before we can assess its precise influence.

In the case of dogs, the position is complicated by the intervention of the vitamin D factor. In the experiments of Weber and Becks a ration poor both in

calcium and vitamin D was fed to puppies weaned at about six weeks, and in experiment for four and a-half to eight and a-half months. Of seven dogs, four survived the experiment and were killed ; but on microscopical examination all showed generalised osteodystrophia fibrosa with tumour-like growths and cysts in the jaws. The authors explain this result as due to combined deficiency of calcium and vitamin D; but although their data do not allow of precise calculations, it is certain that the $CaO : P_2O_5$ ratio must have been very wide, and that it may therefore have played a part in the development of the disease. The authors do not supply information about the state of the parathyroids, so that we have no knowledge as to whether these bodies are affected in the artificially produced disease of this domesticated animal. Although Kintner and Holt make the statement that they found no gross abnormalities in the endocrine glands of their horses, it is doubtful whether the parathyroids were as closely examined as they deserve to be. Our data, from the point of view of comparative pathology, are therefore still fragmentary. Lesions of osteodystrophia fibrosa have been produced in various experimental animals in various ways, and have occurred spontaneously under various ætiological circumstances, in some cases with, and in others apparently without, concomitant changes in the parathyroids. The records show that changes similar to ostitis fibrosa can be produced by nutritional defects, toxic influences, bacterial infections and endocrine disturbances. By some it is thought that the cause operates *via* the parathyroids on the mobilisation of calcium in the bones. By others it has been suggested that the disturbance in mineral metabolism is primary, and that the enlargement of the parathyroids is a secondary phenomenon. But the two views are not necessarily contradictory. It seems safe to assume that if the epithelial bodies, as physiological centres concerned in calcium metabolism, are primarily affected, the disease can declare itself in the absence of nutritional defects. It is equally reasonable to assume that a disturbance of calcium metabolism leading to osteodystrophia fibrosa may follow mineral imbalance, even without changes in the parathyroids. We know that bovine osteomalacia can arise as a " straight aphosphorosis " under natural or artificial conditions, and can be prevented by supplementing with phosphorus and nothing else. We also know that excess CaO in the diet can aggravate the effect of low $P_2O_5$, *i.e.*, when the phosphorus supply is on the border line the addition of chalk to the ration can induce osteomalacia. In the case of horses, it appears that excess phosphorus in some way interferes with the metabolism of calcium, and induces osteodystrophia fibrosa.

In the case of osteodystrophia fibrosa of the horse, we may suppose that the excess phosphorus in the causal diet is absorbed into the blood stream, and that its subsequent elimination imposes a drain on the calcium reserves of the skeleton. Skeletal tissue is absorbed, and then rebuilt on a reduced scale and on a different architectural pattern, which requires only half the calcium in the original structure. The result is a correspondingly light skeleton, for which the reduced calc um resources are still sufficient, and which is more or less completely calcified. But since the new fabric is lighter and more fragile, it is not sufficiently strong to resist

traumatic influences, or the normal stresses and strains of certain functional activities, *e.g.*, the pressure of mastication in the jaws. Here the growth of tissue is stimulated under the influence of constant injury, and this leads to the formation of fibroma-like tumours. As in the case of osteomalacia, there is an attempt to strengthen certain bones, *e.g.*, maxilla, mandibula, ribs, scapula, pelvis, etc., but in the case of osteodystrophia fibrosa it is with a tissue which does not strictly follow the normal pattern, and calcifies more or less completely. In the case of osteomalacia the new tissue is built on a more or less normal pattern, but it is uncalcified.

Why the bovine can tolerate a $CaO : P_2O_5$ ration which induces osteodystrophia fibrosa in the horse, and why the equine escapes osteomalacia on pastures which induce that disease in the bovine, I am unable to explain. The fact remains that typical osteomalacia has not yet been diagnosed in the adult horse on unequivocal histological evidence, and that osteodystrophia fibrosa has not yet been described in cattle. Is it that the physiological mechanism for the conservation of calcium is different in different animals? If that mechanism is under endocrine control, may not the difference lie in functional differences of the endocrine centres? Is it that in the case of the horse we may yet have to call in a concomitant disturbance of the parathyroids, as the result of toxic or infectious agencies, to provide a full explanation for the ætiology of osteodystrophia fibrosa? It is true that we have no experimental evidence for this, as at present all the evidence is in favour of mineral imbalance unalloyed, and our therapeutic and preventive treatment must therefore take the direction of keeping the $CaO : P_2O_5$ balance for the ration at the point experience teaches us to be optimal. But can we be sure that in so preventing and curing the disease we are not still overlooking some significant point in its ætiology? To me, the different reactions of different animals to mineral deficiencies and mineral imbalances suggest the need for further experimental research.

In conclusion, however, I may summarise the facts as I see them with as little speculation as possible:

(1) The osteodystrophic diseases of the domesticated animals, rickets, osteomalacia and osteodystrophia fibrosa, can all be classified as dietary diseases, even if dietary factors are not always the only ones in their ætiology.

(2) They affect the whole skeleton, and arise from a withdrawal of calcium phosphate which cannot be replaced. Pathological changes in the bones are therefore inevitable, but the changes may vary according to age, species of animal and mode of life.

(3) The organism attempts to repair the defects, but does not succeed in restoring the former architecture, since sufficient supplies are not coming in. The result is a structure of inferior quality, usually amounting to a mere makeshift.

(4) The normal functioning of the mineral metabolism of an animal depends upon the harmonious interaction of three dietary factors: vitamin, calcium and phosphorus. The absence of any one of them might be expected to have the

same result in all animals, but in practice this does not seem to be the case. Some species react more readily to one deficiency than another, whilst in other species the same individual may react in two different ways at the same time. Different pathological pictures appear to result from identical causes, and the same pathological picture may be presented by diseases which from the ætiological point of view are different.

(5) The part possibly played by disturbance of endocrine functions, with or without concomitant dietary defects, and the possible significance of differences in endocrine mechanisms controlling mineral metabolism, are as yet unexplored in the domesticated economic animals.

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# THE INTERNAL PARASITES OF DOMESTIC CARNIVORES: A SURVEY.

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Dogs and cats were probably the earliest of all wild animals to be domesticated by man. In spite of numerous attempts to tame other wild carnivores—especially some of the wild cats and the marten cats (with which some partial success was obtained by the Greeks)—only some varieties of the pole-cat (under the name of ferret or fitch) became completely domesticated until relatively recently, when the silver fox was bred in captivity in Canada. There is little doubt now that it also must be accepted as domesticated. Attempts are now being made with

various other wild carnivores—raccoon, skunk, mink and various other mustelids —but it is premature to consider them as being as satisfactorily domesticated as is the fox. As practically nothing is known of the parasites of ferrets, this survey will be mainly confined to dogs, cats and foxes.

The principal internal parasites of carnivores are protozoa and helminths, and it will be more convenient to discuss these according to habitat in the animal.

### The Alimentary Tract.

In the œsophagus of dogs in warmer countries is found the peculiar nematode *Spirocerca sanguinolenta*, which reaches this situation not directly, as might be supposed, but by a migration through the aorta wall from the blood stream. While this worm is present in the Mediterranean region, it is absent from Britain and North America.

In the stomach of the cat in Britain and other parts of Europe is found the tiny nematode *Ollulanus tricuspis*. This trichostrongyle is the smallest parasite found in domestic animals, the mature females being under a millimetre in length. It does not lay eggs, but is viviparous, the living larvæ leaving the host in the vomit. There is some evidence that this species may be able to produce successive generations within the same host; if this proves to be the case, it will be unique among helminths. The majority of the important worms in carnivores live, as adults, in the small intestine. So far as northern temperate climates are concerned, the most important of these are the three species of Ascarids: *Toxascaris leonina*, *Belascaris marginata* (=*Toxocara canis*) and *B. mystax*. The first of these is found in all three hosts, the second in dogs and foxes, and the last in cats and foxes. They have a life-cycle similar to that of the other Ascarid worms, and their larval stages wander through the liver and lungs before taking up their final habitat in the intestine. As adults, they may produce considerable discomfort, and owing to their habit of wandering into the stomach they often cause vomiting. Their most important effects, however, are produced as larvæ. There are innumerable cases of infection of pups with Ascarid worms *in utero*; so much so that some observers regard it as the rule rather than the exception. The eggs hatch in the intestine of the bitch, and migrate in the usual way through the intestinal wall. Instead, however, of passing from the lungs to the trachea, they return to the systemic circulation, and reaching the uterus, migrate into the liver and lungs of the fœtus, remaining there until the pup is born. About five days after birth they descend into the intestine to complete their development in the normal manner. The accumulation of larvæ in the fœtus may be sufficient to cause the death of the pup before, during, or immediately after birth. Quite often the bitch has no adult worms at all in her intestine, all the larvæ she has swallowed being passed to the young, and the cause of the disease is often overlooked. Curiously, this prenatal infection is much more frequently seen with *Belascaris marginata* than with any other species, and it is consequently the commonest form found in pups.

The other important roundworms in the small intestine are the hookworms. In northern temperate climates such as Britain and Canada, only dogs and foxes are infected, the hookworm involved being *Dochmoides* (=*Uncinaria*) *stenocephala*. In the southern parts of Canada, however, as well as in the United States generally, this species is replaced by *Ancylostoma caninum*, which can also infect cats. The hookworms are tissue feeders, but their digestive glands contain an anti-coagulant, and this often permits of a considerable loss of blood from the tissue on which they are feeding. They do not appear to digest this blood, however, and quite considerable quantities pass through their digestive system unchanged. This fact probably accounts for the profound anæmia which accompanies a heavy infection. In North America it has been found that canine and feline races of *A. caninum* exist, and that these are transferred with difficulty from dogs to cats or cats to dogs. A third species of hookworm, *A. braziliense*, is also found in cats and dogs in warm climates. Both species of *Ancylostoma* are quite common in a variety of wild carnivores.

The Trichina worm (*Trichinella spiralis*), although it can live in practically any species of mammal, and occasionally even in birds, and although it is most important economically in pigs, is essentially a parasite of carnivores. The adults live in the small intestine, of course, although the larval stages are found in the voluntary muscle fibres. It is by no means uncommon in wild carnivores such as bears and foxes, but it also occurs in domestic dogs and cats. It appears to do its animal hosts little or no harm, and where carnivores are not eaten it is of little public health importance. In North America and Continental Europe, however, where bears are shot and eaten, it has given rise to a number of epidemics of trichinosis. The parasite is widespread nowadays, although it probably originated in the temperate or sub-arctic regions of the world. While we have no natural records from dogs and cats in either the United Kingdom or Canada, it is definitely known to occur in other animals in both these countries ; in parts of the United States, where systematic examinations have been made, both dogs and cats have been found to be infected.

Among the commonest parasites of the small intestine are the tapeworms. In town dogs and cats the form most frequently encountered is *Dipylidium caninum*, and there is some evidence to suggest that this species is becoming differentiated into biological strains peculiar to dogs and to cats. It is also found in foxes, but much less commonly. Its urban distribution is explained by the fact that its intermediate hosts are biting lice and flea larvæ. Cats, both in town and country, harbour a species of *Tænia* (*Tænia tæniæformis* = *T. crassicollis*), which is carried by mice and other small rodents (including muskrats, but not hares and rabbits) ; this tapeworm is also found in many species of wild cats. The other tapeworms of carnivores are most common in country animals. A species of *Mesocestoides*—there is still some doubt about its name—occurs in wild foxes and badgers in Britain, but it has yet to be recorded from dogs, cats and silver foxes in the United Kingdom and Canada, although it is found in

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these hosts elsewhere. This tapeworm is characterised by a centrally-placed median genital pore. It superficially resembles *Dipylidium*, but as that species has a genital pore on each lateral margin, it can be easily distinguished from it. Its larval stage is carried by various small mammals and reptiles.

In dogs, in Canada, but apparently not in England and Scotland, is found another tapeworm with a centrally placed median genital pore. This is *Diphyllobothrium latum*, often called the broad tapeworm. It occurs also in man, both in Europe and North America, but appears to be absent from the British Isles, except Ireland. It is a large tapeworm involving two successive intermediate hosts: a fresh-water crustacean and a fresh-water fish. In Canada it is harboured by a variety of wild carnivores as well as by dogs.

The most frequent tapeworms of dogs and foxes, however, are the various species of *Tænia*. *Tænia pisiformis* (=*T. serrata*), which has its larval stage in hares and rabbits, is probably the most common in the United Kingdom, closely followed by *T. hydatigena* (=*T. marginata*), the larval stage of which is *Cysticercus tenuicollis* so constantly seen in sheep and pigs ; the latter species is probably the form most usually seen in Canada.

*Tænia ovis*, which has its larval stage in the muscles of sheep, is common only in Western Canada and the United States, although it is occasionally seen in Britain and elsewhere. *Tænia serialis* and *Tænia multiceps*, which have a cœnurus instead of a cysticercus as a larval stage, and which may actually be the same species, are much less common nowadays than they used to be. The second species, however, is still not infrequently seen in Wales and the Scottish Highlands. It is, of course, the cause of " gid " in sheep and, less frequently, in calves. A few years ago it completely wiped out a herd of sheep in Western Canada, the epizoötic being traced to a single dog. These tapeworms as adults are relatively harmless parasites, and their importance lies in the serious effect they often produce in their intermediate hosts.

The last and most important of all the tapeworms of carnivores is *Echinococcus*—a genus which probably contains several species. The most important form in northern climates is *E. granulosus*. In Britain this parasite is found naturally in wild foxes and in dogs, but as it generally is larger in the fox than the dog, it may be assumed that the former is the more natural host. It can be transmitted to cats, but only with difficulty, and these animals are not important hosts. Owing to its small size it is seldom seen, but its larval stage, the hydatid cyst, is only too common. The parasite is practically cosmopolitan, and as hydatid cysts infect all species of mammal, from man to marsupial, it is of the greatest economic importance. While horses, pigs and cattle are commonly infected, sheep appear to be the optimum intermediate hosts, and the disease is commonest where the sheep population is most dense. Both the adult tapeworm and the hydatid cyst occur in a variety of wild animals, thus making eradication difficult ; but a considerable degree of control can be exercised by medicinal treatment of dogs for tapeworms (although destruction of any animal known to

harbour the adult is strongly advised), and proper disposal, by heat, of all infected offal.

Hydatid infection in man is almost always contracted in youth, although owing to the slow development of the cyst, the disease does not occur until much later. This fact is seldom taken into account in attempting to trace the origin of the infection in the occasional fatal case seen in Britain. The owner's present pet dog is seldom, if ever, the source of the cyst : it should be looked for some distance in the past history of the patient, and will usually be traced to unwashed hands or green food on a farm in sheep country, perhaps twenty years earlier. Human prophylaxis depends on clean eating habits and well-washed garden produce which is to be eaten uncooked ; water is a negligible source of infection, as the eggs sink into the mud at the bottom of ponds.

The salmon-poisoning fluke—*Troglotrema salmincola*—occurs in the small intestine of dogs in Western Canada and the United States. It is a minute parasite, the second intermediate host of which is a fish—usually a salmon ; the first is, as usual, a snail. The parasite is widely distributed in Canada in wild carnivores, and there is some evidence to suggest that the disease in Western dogs is caused by a virus which is transmitted by the fluke. This point, however, requires further investigation.

Numerous heterophyed flukes occur in dogs and cats in various parts of the world, but they are very small forms doing apparently little or no damage.

The only important protozoa of the intestine of carnivores are various coccidial parasites of the genus *Isospora*. They are very common, but just how frequently they cause disease is unknown ; the common opinion is that, generally, in otherwise healthy animals, they are not very pathogenic. The other genus of coccidian parasites of mammals—*Eimeria*—is rare in carnivores. It has been recorded from dogs in Europe, and we have found *Eimeria* cysts in the fæces of Canadian dogs, without, however, being able to rule out the possibility of their being pseudo-parasites.

The only parasite of the large intestine is the whipworm, and while it is widely distributed, it seldom is found in numbers, and probably is of little importance.

The liver is a favourite site for the development of flukes. I have on one occasion found *Fasciola* in the liver of a dog in Britain, but this is the only record available. In Canada and elsewhere, however, various fish-transmitted flukes of the family Opisthorchidæ (chiefly belonging to the genera *Parametorchis* and *Opisthorchis*) are found in dogs and cats, and there is reason to believe that they are by no means uncommon in Northern dogs. As the dog there is of paramount importance as a transport animal, they are of considerable economic significance in Arctic and sub-Arctic regions.

### The Respiratory Tract.

In the nasal cavity of dogs occurs a large degenerate arachnid parasite called *Linguatula serrata*, the larval stages of which are commonly seen in the lymph

glands, liver and lungs of cattle, sheep, pigs and other animals. While there are few records of the adult parasite from both Britain and North America, it is probably much more common than is generally believed. Its full life cycle is still unknown.

In the trachea of dogs occur nodules produced by the nematode *Oslerus osleri*. First found in Montreal by Osler, this parasite has been reported from many parts of the world, including England. Unfortunately little is known about either the worm, its life history or pathogenicity, but obviously a heavy infection would mechanically interfere with breathing.

The "lung worm" of the dog—*Angiostrongylus vasorum*—while common in Southern Europe, has not yet been recorded in Britain or North America. The adults live in the pulmonary artery, their eggs being carried to the lungs, where they hatch and produce a crop of pseudo-tubercules. Its life cycle is unknown.

The "lung worm" of the cat—*Ælurostrongylus abstrusus*—has a similar habitat, but a wider distribution. It occurs not only in Europe, but in the United Kingdom and the United States, although it has not yet been recorded from Canada. It is carried from cat to cat by mice, which act as intermediate hosts. Its developing eggs create a considerable host reaction, which, however, quickly subsides, and unless a heavy infection is present no symptoms are produced. A considerable number of larvæ developing simultaneously, however, is too big a strain on the lungs, and pneumonia results. No method of treatment is known, and prevention is obviously impracticable.

Two species of lungworms are found in foxes, *Capillaria aerophilia* and *Crenosomia semiartmata*, and while both forms occur in foxes, both in Canada and Britain, the former appears to be the more common. Its life-cycle outside of the body is direct: inside the host it is still incompletely known, but it almost certainly migrates to the lungs *via* the lymphatic system. Infection is by swallowing the embryonated egg from the feet or the fur, or in contaminated food, and in parts of Canada the resulting infections have been so heavy that many fox breeders have been put out of business. The worms live in the bronchi and bronchioles, causing a catarrhal bronchitis, and may actually occlude the lumen. Fox pups are especially affected, and no satisfactory medicinal treatment has yet been evolved. Fortunately, an effective preventive system has been worked out in Canada, and the disease is now largely under control. This system consists in keeping the young foxes on raised board or wire floors and under cover. While it is admitted that a natural life in earth dens would probably produce finer pelts, it is also realised that artificial fox raising is unnatural and that its practice must be attended by some modifications from a strictly "normal" life, to avoid disease caused by confinement on a limited space.

Little is known of *Crenosoma semiarmata*, which, as a rule, is confined to the smaller air tubes. It appears to affect the lungs in much the same way as does *Capillaria aerophila*, but its life cycle is quite unknown.

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### The Urinary Tract.

The kidneys and bladder of dogs and cats are free from parasites in Britain, but in Canada a large earthworm-like roundworm, called *Dioctophyme renalis*, sometimes occurs in the kidneys of dogs and foxes, although its normal host appears to be the mink. It not only completely destroys the kidney substance, but may escape into the peritoneal cavity. Its life history is unknown, but it is believed to be carried by fish. In the bladder of foxes, both in Britain and in America, is often found a very small nematode called *Capillaria plica*. It occasionally is found also in dogs. If present in numbers it may cause a considerable degree of cystitis.

### The Blood Stream.

The only important helminth of the blood stream of dogs (excluding the so-called "lung-worm") is the "heartworm," *Dirofilaria immitis*, which, although absent from Britain and Canada, is common in most warm countries, including the southern United States. Although this parasite is of considerable size, and may be present in numbers, it often shows no symptoms of its presence until the dog is undergoing exercise. The worms then may be mechanically moved forward to completely occlude the aorta; the animal drops down as though dead, but quickly recovers as the worms move back to the heart again. Un doubtedly, however, the worms sometimes do kill the dog. The larvæ are passed into the blood stream and the vectors are various species of mosquitoes. "Fouadin"—an antimony preparation in use for human bilharziasis—has recently been found to give good results with this disease also.

While quite a number of protozoan parasites have been reported from the blood of dogs—including various Trypanosomes and *Leishmania*—the most important are the piroplasms, the cause of malignant jaundice. Babesias have been known in dogs for nearly forty years, but it is still uncertain how many species exist. The commonest is undoubtedly *B. canis*, which occurs in Southern Europe, Asia and Africa, but is absent from Britain and North America. In South America piroplasms have been recovered from dogs; while some authorities believe that these belong to a different species; others are not yet satisfied that this parasite is not *B. canis*. The Babesias are all tick carried, and their distribution is to a large extent governed by the distribution of the vectors. It is important to note, however, that *Dermacentor venustus* in North America has been proved capable of carrying the infection, although it has never gained a foothold in that continent.

There are no important blood parasites in foxes or in cats (other than the "lung worm," which is discussed elsewhere).

### General.

Domestic carnivores occupy an almost unique position among animals. Dogs and cats are kept in much more intimate contact with human beings than are other animals, and many of their diseases and parasites are transmissible

to man, and some of these are among the most serious, affecting mankind and domestic herbivores and pig. The parasitic diseases of these animals are at least as important as their bacterial diseases, and, with the conquest of distemper, promise soon to occupy first place. They are equally serious in foxes. Fortunately, medicinal treatment is available for many of these conditions—although by no means for all. We have a fairly detailed knowledge of the bionomics, life histories and methods of infection of practically all the important forms, and efficient means of prevention can be employed in many cases.

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# THE COMPOSITION OF "WITCH'S" MILK.

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MILK secreted by human infants and other immature mammals is commonly referred to as " Witch's " milk (Hexenmilch) and has been the subject of frequent comment in the professional journals. In 1847, John Coventry, surgeon, in a letter to *The Lancet*,[1] records the case of a female infant three weeks old with mammary glands the size of a walnut from which there issued a continuous oozing of milk. In the same periodical,[2] in 1867, was published a considerable correspondence from medical practitioners who gave instances of the secretion of what appeared to be normal milk from the mammary glands of both male and female children at birth, or during the first few days after birth. In none of these early cases was the fluid analysed.

As examples of the occurrence of " Witch's " milk in the lower mammals may be quoted those reported by Ammon and Dayot. Ammon[3] records the mammary secretion in a five-weeks-old foal, and states that it contained : Casein, 0.5 per cent. ; albumin, 1.02 per cent. ; extractives and sugar, 3.67 per cent. ; and ash, 0.44 per cent. Dayot[4] relates his experience of a colt, eleven days old, which " could be milked like a cow."

During the course of an investigation into the composition of mare's milk,[5] two instances of foals secreting milk at birth were reported, and the milk was sent for analysis.

*Case No.* 1.—During the foaling season of 1928, some milk-like fluid was drawn from a filly foal of a Clydesdale mare. The foal, when two days old, had a swollen and painful mammary gland from which about half a pint of milk was withdrawn by hand milking. This, and a sample of milk from the mother, were analysed, and found to have the following percentage composition :

| | Total Solids | Protein | Fat | Lactose | Ash |
|---|---|---|---|---|---|
| Mother ... ... ... | 11.43 | 3.23 | 0.67 | 6.99 | 0.54 |
| Foal ... ... ... | 8.64 | 2.96 | 0.10 | 5.21 | 0.37 |

In contradistinction to Case No. 2, all the previous foals, seven in number, born of this mare were normal.

*Case No.* 2.—In 1929 a sample of milk from a foal four days old was received at the laboratory. The dam of the foal had an interesting breeding record. She was a cart mare and had had three foals. The first foal had a cleft palate and supernumerary digits; the second, born in 1928, died shortly after birth; the present (third) foal was born with a remarkably thick umbilical cord and abnormal mammary glands, about the size of a large teacup and very hard. The percentage composition of the milk from the foal and the dam was as follows:

| | Total Solids | Protein | Fat | Lactose | Ash |
|---|---|---|---|---|---|
| Mother ... ... ... | 10.27 | 2.72 | 1.30 | 5.71 | 0.54 |
| Foal ... ... ... | 10.18 | 1.76 | 2.12 | 5.91 | 0.39 |

Four days later, when the foal was eight days old, a further sample was obtained, and the total protein was separated out to its casein, globulin and albumin constituents. This sample was found to contain 3.58 per cent. of protein, made up of casein, 2.03 per cent.; globulin, 0.86 per cent.; and albumin, 0.69 per cent. The higher percentage of protein in the later sample is probably accounted for by concentration in the udder resulting from incomplete milking, and the relatively large amount of globulin seems to suggest that the milk was of a colostral nature. Nevertheless, in view of the well-known difficulty of accurately determining the relative proportions of the milk-protein constituents —a difficulty that is even greater with mare's milk than with cow's milk— absolute accuracy is not claimed for these results.

In view of the fact that apparently no analyses have been made of the ash of foal's milk, a portion (20 grammes) of the second sample was utilised for this purpose, and was found to contain 0.58 per cent. of ash, an increase over that obtained (0.39 per cent.) in the first sample. Of mineral constituents it was only possible to determine the percentages of chlorine, calcium, magnesium and phosphorus in this sample; and, unfortunately, no further samples were obtainable. The ash was found to contain chlorine, 0.230 per cent.; calcium oxide, 0.041 per cent.; magnesium oxide, 0.014 per cent.; and phosphoric anhydride, 0.079 per cent. The chlorine quantity is considerably greater than that found in normal mare's milk; while the calcium is less than the normal, and the magnesium and phosphorus are approximately the same.

In more recent work at present in progress, it has been found that the mineral content of mare's milk gradually decreases from parturition to the weaning. Thus in one mare (Shetland pony), where weekly samples were taken throughout the lactation period, the percentage fell from 0.57 per cent. to 0.19 per cent. 161 days after foaling. During weaning, when milk collects in the mammary gland, the percentage of ash is greatly increased; thus in the pony mare referred to the ash content rose from 0.19 per cent. to 0.67 per cent. at weaning. This increase, the nature of which is still being studied, was found to be due mainly to chloride, which rose from 0.021 per cent. when the total ash was 0.19 per cent., to 0.180 per cent. when the ash rose to 0.67 per cent. This second sample is, therefore, milk from a turgid udder, which explains the difference found in the two samples.

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The foregoing analyses clearly suggest that the mammary secretion of the newborn is truly milk; it has, however, not yet been determined if so-called "Witch's" milk is chemically and physically normal milk or to what extent it has the characteristics of colostrum. As already stated, the amount of globulin found in one of the samples is greater than that found in normal mare's milk, and is therefore suggestive of colostrum. To those interested in the study of the causation of milk secretion the production of such milk should be of particular interest.

Numerous cases have also been placed on record of adult animals secreting milk at abnormal periods. For example, Monvoisin[7] refers to abnormal secretion in a mare, a sow, a mule and a heifer, and discusses the well-known and common occurrence of bitches secreting milk after a period of sexual activity. He gives Wedemeyer's analysis of such milk from a ten months' old bitch as follows: Total solids, 31.91 per cent.; protein, 10.18 per cent.; fat, 15.54 per cent.; sugar, 3.71 per cent.; and ash, 1.10 per cent. This is somewhat richer in protein, fat and total solids than normal bitch's milk, but supports the contention—disputed by some—that the fluid secreted under these circumstances has the characteristics of normal milk.

Grimmer[8] refers to what he calls "Witch's" milk at some length, pointing out its somewhat frequent occurrence and giving numerous references to published papers. Among these, special attention is directed to an early record by Dujès,[9] who analysed the milk secreted by a mule and found its composition to be: Protein, 1.95 per cent.; fat, 1.7 per cent.; sugar, 5.13 per cent.; and ash, 0.38 per cent.; from which it is clear that the fluid secreted by this particular lactating mule had the same composition as normal mare's milk.

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# PUPPY FEEDING: SOME OBSERVATIONS ON RECENT RESEARCH.

By A. GREEN, M.C., Ph.D., M.Sc., F.I.C.

*Chief Chemist to Messrs. Spillers, Limited.*

On a previous occasion[1] an outline has been given of the fundamental nutritional principles which are used to guide the manufacture of modern dog foods, and it is now proposed to indicate how some recent researches affect proved methods of feeding and rearing puppies.

The first essential is the correct pre-natal feeding of the bitch, and in this connection the extensive researches of Mrs. Mellanby[2] are of particular interest. The ultimate object of Mrs. Mellanby's work was the control of dental caries in man, but it involved a detailed investigation of the relationship between dental structure and diet in dogs. The close connection between dental disease and general health is becoming increasingly evident, and Mrs. Mellanby has said that " of all the diseases which beset humanity, it is probable that dental diseases are responsible, directly or indirectly, for a larger aggregate of ill health and unhappiness than any other form of disease " ; and this applies to the dog as well. Mrs. Mellanby's work with dogs showed that the feeding of the mother during pregnancy and lactation affects the permanent as well as the deciduous teeth of the offspring. It had been previously demonstrated that the maternal feeding was of vital importance in bone formation and in the incidence of rickets ; and what is true for the teeth and the bones is true also for the whole of the young organism.

It is well established that from conception onwards the nutrition of the dam must receive the closest attention in order both to ensure the healthy growth of the developing embryos and also to maintain the bitch herself in sound condition ; for if her food is not adequate in quality and quantity then the embryonic structures will grow at the expense of the mother's organism. The age-long recognition of this truth is embodied in the old saw : " a tooth for every child." Fortunately this is no longer true if the mother's diet contains adequate minerals, and also the vitamins to ensure their proper utilisation.

The food of the puppy is supplied by the bitch from conception to weaning, and during this period her diet should contain liberal, but not excessive, quantities of first class protein, adequate minerals properly balanced, and also the essential vitamins. This group of nutritional factors has a dominating influence on the quality and quantity of the milk supply, and to fulfil these requirements Spillers have put together certain ingredients under the name of " Saval" which are of particular value. This is primarily a food for puppies after weaning, for it contains all the nutritional elements required by a rapidly-growing young animal ; but it is also of the greatest value to the bitch and therefore to her litter both before and after parturition.

The nutritive ratio of " Saval " is approximately 1 : 3. The recent work of Lössl[3] indicates that this is a satisfactory average value for young dogs ; but especially for the larger breeds a narrower ratio can be obtained by feeding a small supplement of minced fresh meat, preferably raw. " Saval " is a meal constituted according to the principles indicated above ; its protein ingredients are of the highest biological value and are selected in the light of the work the food is intended to do ; and the iodised mineral supplements are balanced mainly with a view to sound bone and teeth formation and preservation. The vitamin carriers are added under strict control, and their unimpaired potency in the foods after manufacture is checked by analytical and biological feeding tests.

XUM

Another aspect of Mrs. Mellanby's researches is of considerable general importance in connection with dog feeding. This work indicated that in the case of dogs who were receiving little or none of the calcifying factor vitamin D, such cereal products as oatmeal, wheat flour and wheat germ display an anti-calcifying tendency and defective dentition results. This effect, however, is entirely counteracted and overcome when the diet contains vitamin D, and under these conditions, to quote Mrs. Mellanby, "the teeth are perfectly formed." Thus the possibility of dog foods with a cereal base giving rise to defective teeth and bone formation does not arise if these foods are adequately supplemented with vitamin D in the form of cod-liver oil; and the proved value of biscuits and biscuit meals is in no way diminished by Mrs. Mellanby's findings, provided these foods contain adequate amounts of easily assimilated minerals and also the vitamins to ensure their economical utilisation by the organism.

In all Spillers dog foods the vitamin and mineral levels are high, and the cereal ingredients are freshly milled. Wheat germ is used in conjunction with active yeast as a carrier of the vitamin B complex, which is closely related to the correct functioning of the nervous system. Immediately after manufacture in Spillers' own mills this wheat germ is so treated that the toxic elements, which are sometimes found in the stale product, have no opportunity to develop, and this treatment leaves the vitamin potency unimpaired.

It can be said that the researches mentioned above afford further confirmation of many years' practical experience as to the value of a cereal basis in dog feeding; but it must not be forgotten that this cereal basis must be fully supplemented by the other factors required for adequate nutrition, and these factors should always be looked for in any manufactured dog food.

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# Clinical Articles

## Fits Following the Ingestion of Straw and Hay.

By R. HUDSON, F.R.C.V.S.

*Retford.*

THE sudden death of one or more pigs in a lot is a common occurrence, and mostly due to some dietetic cause. "They were alright when fed at night, and dead the next morning" is the usual remark. If pigs have been used to bedding from birth, there is little risk in continuing it, but if not, there may be trouble

when it is supplied. Sometimes a change from wheat to barley straw or hay will give rise to fits. Recently an owner had kept six three-months-old pigs on the bare floor, and thinking that they would be more comfortable if bedded, he put them on straw. The following morning one pig was dead, one dying in a fit, and three others were wandering about silly. The feeding had been the same for several weeks, and no fresh meals had been obtained. The food consisted of sharps or seconds in water.

The dying pig was killed and dressed, and the post-mortem examination of it and the dead pig showed engorgment of the stomachs with straw, and blockage of the pylorus with plugs of it. It appears that the sealing of the pyloric valve with a plug of straw is the cause of the fits, for in several cases the stomach had contained very little. If there is a blockage, and food, particularly food of a fermenting nature such as potatoes, mangolds, etc., is taken, then the end may come quickly. The remaining pigs were given a powder of calomel, jalap and mag. sulph. added to very thin food for a few days. They recovered.

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## Casting and Securing of Large Pigs.

By R. HUDSON, F.R.C.V.S.
*Retford.*

GOOD'S method of pulling up the nose and chloroforming is excellent, but one does not always require a general anæsthetic. The placing on its side, and the holding there of a 15 to 25 stone pig is not easy without ropes, so the following procedure is adopted: Three ropes are required, thin halter shanks will do. One is placed round the neck and tied light enough to prevent the shoulders from pulling through. The others are looped round the hind legs below the hocks. One of them is passed through the neck rope, the hind leg pulled off the ground, and the rope tied; then the other likewise. To prevent the pig from wandering about, she should be tied by the snout to a post or manger ring.

# Abstracts of Current Literature

**Pigeon's Milk.** By L. BINET and M. BUREAU. *La Presse médicale*, May 27th, 1933; *Rev. Gen. Méd. Vét.*, July, 1933.

IN his volume of collected works Buffon refers to the life and habits of the pigeon, and does not fail to record the active participation of the male in all the business of family life. "The male likes to partake of and busy himself in maternal cares, and regularly takes his turn in sitting on the eggs and young." To-day we know he does more than this. In pigeons the male and female each secrete a kind of "milk" in their crop which suffices for the nourishment of

newly-hatched pigeons. J. Hunter (1786) seems to have been the first man to note this phenomenon. Claude Bernard (1859) in one of his lessons enlarges on this curious point of comparative physiology. "The secretion of the crop of pigeons is analagous to the lacteal secretion of the mammiferæ. This secretion commences in the cock and hen pigeon before hatching, and lasts for some days after. At this time a true glandular organ is formed in the crop of the pigeon; whilst the external muscular part does not react, the mucous membrane of the two lateral pouches of the crop undergoes a considerable hypertrophy, and presents a convoluted surface similar to that on the outside surface of the brain. The vessels of the crop themselves increase in volume. It is in the lateral pouches of the crop, at the surface of the hypertrophied mucosa, that one finds a kind of white substance, analagous to coagulated milk. Father and mother pigeons regurgitate this substance, and allow a beakful of it to be absorbed by their young during the first days following hatching." Since the fundamental researches of Hunter and Bernard, many biologists have called attention to the matter of pigeon's milk. The secretion of the milk takes place longer than Bernard thought, *i.e.*, for eight days. Charbonelle, Salle and Phisalix observe that it commences on the eighteenth day of the sitting, and finishes the twentieth day after hatching. The product elaborated by the crop is a whitish mass, a true "milky pap." Chemists have published an analysis of it. Fats and proteins are present in large quantities. Mineral salts are present, and some ferments, amylose and saccharose in periods of repose. Histophysiology shows that a process of desquamation of the epithelial cells of the hypertrophied mucosa of the crop takes place, and that these cells contain large quantities of fat. The fat globules are secreted by these cells in the period of incubation of the egg, just like fat globules in the epithelial cells of the mammary gland. The pigeon doubles its weight at birth in forty-eight hours. In twenty days it may increase in weight from 20 grammes to 435 grammes (three-quarters of an ounce to fourteen and a-half ounces). Pigeon's milk much resembles the milk of the rabbit in its contents of proteins and fats. The pigeon and rabbit have the quickest growth in their respective spheres.

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**Types of Clostridium welchii Found in Dysentery in Lambs.** By H. Marsh, E. A. Tunnicliff and E. Junghew. *Journ. Infect. Dis.*, Vol. 51. Pp. 330–335.

This article deals with the study of an acute fatal diarrhœa of young lambs in the intestine of which *C. welchii* was found constantly present. As the disease corresponded somewhat in its clinical aspect with lamb dysentery as found in England, and with the cause of which a variety of the *welchii* group is closely associated, the authors decided to study the strains of the organism they isolated. They had obtained cultures of the English lamb-dysentery type of *C. welchii*, and included this culture in their programme, as well as cultures of *C .welchii* obtained from the intestine of normal lambs. The result of their investigation

was that " in both normal lambs and lambs dying with dysentery, the types of *C. welchii* present are the ordinary types 1 and 2, and that culturally they do not show any of the differential characteristics described for a variety of *C. welchii* that has been incriminated as the cause of dysentery in lambs in England."

# Obituary

## VETERINARY-COLONEL SIR FRANCIS DUCK, F.R.C.V.S., and FRANCIS HENRY RIDLER, M.R.C.V.S.

By the deaths of Sir Francis Duck and Mr. Francis Ridler, at the ages of eighty-eight and ninety-one respectively, the English veterinary profession loses two of its oldest landmarks. Each man made his mark in his own sphere: the one in the military and other in the civilian side of veterinary life, and each commanded the respect and esteem of his professional colleagues. The London College is proud to have been able to claim them as two of its sons, and the name of Colonel Duck will be remembered by numerous old graduates for the part he played for many years as one of the examiners in the final year for the M.R.C.V.S. Diploma. Always courteous and with a kindly manner which put even the most nervous student at his ease, it need hardly be said that when by the exigencies of Army service he was compelled to resign his examinership there was universal regret on the part of those who had hoped to obtain their Diplomas through his recommendation.

Francis Ridler, on the other hand, represented the best type of general practitioner. Graduating in 1864, he first went as assistant to the late W. Ellis, a large horse practitioner in Liverpool, returning to London in 1866 to commence practice on his own account in Kensington. With his services commenced the romance of the great commercial house of John Barker & Co., for he and the late Sir John Barker were young men together, the latter being at that time a shopwalker at Whiteley's; and it was always a proud boast of Mr. Ridler that " he bought John Barker's first horse." His practice grew speedily, for his honesty of purpose and straightforwardness in all opinions which he gave were proverbial amongst his clients, and his *clientèle* numbered some of the chief commercial firms in the West End of London.

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